PITFALLS IN THE MANAGEMENT OF PERIPHERAL NERVE INJURIES*

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We both create and inherit the pitfalls in the management of our patients. In my practice, there are a few outstanding pitfalls related to traumatic peripheral nerve injuries. Let me share just three pitfalls with you.

The common decision I must make is the indication to re-explore a peripheral nerve lesion that has been sutured elsewhere by a surgeon unknown to me. One has the option to delay exploration until sufficient time has passed to be certain that regeneration has not reached the most proximal motor end plate. To wait always for regeneration to be proved is dangerous, especially in a proximal (high) lesion because in time there is fibrous distortion of the distal receptors, and in time there is decreased axon regeneration. This is pertinent when there is a long distance from the suture line to the most proximal receptors, and the initial suture is inadequate. It is possible to anticipate regeneration by an advancing Tinel's sign or electromyography to measure the electrical activity of muscle. Unfortunately, neither technique will predict the return of function.

During World War II studies, more than 50% of patients who had a nerve sutured and showed an advancing Tinel's sign eventually required a further resection and suture of the original repair. Because there is no reliable technique to provide precise information concerning nerve regeneration, I usually re-explore the referred nonfunctional repair somewhere between three to four months after injury.

When the suture site is explored, the surgeon observes a neuroma-in-continuity. One looks at the lesion and palpates it, with little useful

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information for a decision to resect the suture line or leave it alone. One should stimulate proximal to the suture line to observe distal muscle twitch. The best available technique to resolve this pitfall is a recording of the nerve-action potential by stimulating the nerve proximal to the suture line and recording directly from the nerve distal to the suture line. If a nerve-action potential can be recorded and it conducts more than 40 meters per second, this early lesion is better left alone.*

The second pitfall I experience is the observation of a patient with pain until there is a fixed clinical pain syndrome. It takes five to eight weeks for the osteoporosis of Sudek's atrophy to be obvious on the roentgenogram, and all too often there has been no pain-management program initiated for the patient. The prevention of a clinical syndrome secondary to pain is an important aspect of post-traumatic treatment: avoid edema by elevation, judicious compressive dressings, venous repair in extensive injuries, and steroids in potential problems such as rattlesnake bites; consider appropriate medication such as aspirin to block prostaglandins from injured cells and a mild tranquilizer for the susceptible personality; and be suspicious of prolonged pain.

The treatment of clinical syndromes related to pain has only two principles: relieve the discomfort and institute *active* use of the involved extremity. We concentrate on the relief of pain, and the pitfall is the failure to emphasize the function of the extremity.

Pain syndromes can be evaluated under four categories: peripheral nerve involvement, sympathetic dystrophies, inflammatory pain and stiffness, and causalgia. Peripheral nerve pain can be eased by neuromodulation through transcutaneous electrical stimulation, peripheral nerve anesthetic blocks, and surgical transfer of neuromata. Sympathetic dystrophies secondary to vascular constriction can be evaluated by an ice-water test and treated with a local acting vasodilator such as priscoline. Other sympathetic dystrophies can be treated with a series of central chemical blocks of the sympathetic chain. Inflammatory pain and joint stiffness can be eased with an intravenous regional block with steroids. Causalgia demands a surgical sympathectomy.

The second principle in treatment is encouraged by the surgeon, but done by the patient. Passive physical modalities include: contrast tempera-

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ture baths, massage, ultrasound, and the Jobst splint. Active physical modalities include: repetitive exercises, diversional activities, and functional use of the involved extremity.

The third pitfall has been the incomplete diagnosis of the patient with the double level compression syndrome such as a cubital tunnel syndrome and a Guyon's tunnel syndrome in the same extremity. The surgeon must complete a differential diagnosis for potential compression from the spinal cord to the intrinsic muscles of the hand. Standard physical findings may not define the "double crush" entrapment, and radiographic and electrodiagnostic studies should always be done. Roentgenographic evaluation must include the cervical spine, forearm, cubital tunnel, and carpal tunnel views. Electromyographic studies are useful, but not as valuable as the conduction velocity determination. Usually both motor and sensory conductions are determined. However, several factors can affect conduction velocity readings: age of the patient, temperature of the room (a cold patient has a slower conduction velocity), and technical errors such as recording distance or the position of the extremity. Often, improvement in most of the patient's symptoms can be achieved when one of the two compression levels is released; then the surgeon is faced with the pitfall of reoperating on the decompressed lesion instead of diagnosing the second compression level.